rates, the effects of barbiturates can last for 3 to 4 days. During that time, patients require mechanical ventilation and cardiovascular support in an intensive care unit, precluding adequate neurologic examination. Further, the complications of prolonged coma, such as pneumonia and deep venous thrombosis, are dangers. Finally, the cost associated with barbiturate therapy is considerable. Consequently, the use of barbiturates for the purpose of brain protection is limited.

In patients undergoing clipping of cerebral aneurysms, a barbiturate coma is used only with clearcut evidence of cerebral ischemia during temporary vascular clipping. In this situation, the surface electrocorticogram is monitored in the region of the brain that is considered to be at risk. Upon temporary clipping, if there is evidence of ischemia, the clip is removed and barbiturates are administered. Cardiovascular support is provided by either phenylephrine or dopamine administration. Pentobarbital is administered until a burst-suppression pattern is seen on the surface electrocorticogram. The temporary clip is then reapplied. The total dose required is about 1.0–2.5 g/kg.

Barbiturate coma is induced prophylactically in patients undergoing arteriovenous malformation (AVM) resection in whom the risk of perfusion pressure breakthrough is considered to be high. These generally tend to be large or deep-seated AVMs and lesions with considerable shunting. Prophylactic barbiturate coma in these patients provides excellent operating conditions (brain relaxation and presumably brain protection). In addition, barbiturate coma is also employed intraoperatively in patients who develop intracranial hemorrhage or perfusion pressure breakthrough. There are, however, no data to support the premise that the administration of barbiturates reduces the incidence of perfusion pressure breakthrough. Nonetheless, uncontrolled series have shown excellent outcomes after AVM resections in patients who have developed intracranial hemorrhage or in whom intracranial hypertension has occurred in the postoperative period.

In general, most patients in whom barbiturate coma has been employed can be weaned off mechanical ventilation within two or three days of surgery.

Etomidate. Theoretically, etomidate appears to be the ideal neuroprotective agent. It can reduce $CMRO_2$ by up to 50% by producing EEG burst suppression. Furthermore, unlike barbiturates, etomidate clears rapidly and does not cause myocardial depression or hypotension. Initial studies in the setting of global ischemia demonstrated that etomidate can reduce ischemic injury. The reduction in injury was relatively small, however, and was confined to single structure (the hippocampus). Subsequent studies in models of focal ischemia revealed, surprisingly, that etomidate actually increased the volume of brain infarction. This injury-enhancing effect has been attributed to its ability to reduce nitric oxide levels in ischemic brain tissue (either by inhibiting nitric oxide synthase or by directly scavenging nitric oxide). Since nitric oxide is thought to be important in maintaining blood flow during ischemia, it is conceivable that etomidate might increase the severity of ischemia. The available data therefore do not support the use of etomidate as a neuroprotective agent.

PIYUSH M. PATEL, MD
HOI SANG U, MD
San Diego, California

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Recent Advances in the Management of Spinal Cord Injury

SPINAL CORD INJURY (SCI), a devastating problem that disproportionately affects young males, results in huge costs to patients and society. Though the incidence in the United States has diminished somewhat recently from 50 to 35 spinal cord injuries per million population per year, currently there are an estimated 250,000 American survivors. The resultant morbidity immediately, dramatically, and often permanently changes the survivor's lifestyle and occupation.

Complete spinal cord injury is a total and permanent loss of all neurological function below the level of injury. Incomplete injuries manifest various degrees of distal function in patterns consistent with the neuroanatomical topography of the cord. These include the Brown Sequard Syndrome of cord hemisection, central cord syndrome often seen with cervical hyperextension injuries, and others. Some patients with incomplete SCI have a reasonable chance for functional recovery. Any significant neurological improvement in such patients—by even one dermatomal level—improves quality of life, particularly for injuries in the lower cervical spinal cord.

In dealing with SCI, one must always consider the dual nature of the injury. The neural elements and the musculoskeletal components (the spinal axis) are affected; assessment and management must address both. Diagnosis of the cord injury is made clinically on the physical examination of the patient, whereas its etiology, the bony or ligamentous injury, is identified radiographically.

As soon as any neurological deficit consistent with spinal cord injury is identified, the spinal axis is presumed to be unstable until proven otherwise, and full spinal precautions must be instituted to prevent further harm. The patient is immobilized in a rigid cervical collar secured to a full-length backboard with straps securing the torso and pelvis. Any patient transfers or movements must preserve this alignment by using concerted effort (four-person log-rolling technique).

In 15% of cases with one spinal cord fracture, a sec-

ond noncontiguous fracture exists. Screening evaluation of the entire axis from occiput to sacrum is necessary and should be done rapidly so that the backboard can be removed to prevent skin decubiti. Identified fractures require further delineation with computed tomography or magnetic resonance imaging. Once mechanical stability has been assessed, treatment of the spinal axis can be planned. Treatment decisions include restoring stability, preventing delayed deformity, immobilization versus surgery, and the timing of surgery.

There is currently no cure for the neurological deficits incurred with spinal cord injury. Medical management of the patient focuses on avoiding secondary injury and rapidly instituting methylprednisolone therapy as appropriate. Adequate resuscitation of the patient begins the process of restoring homeostasis. Hypotension and hypoxia must be avoided scrupulously and treated aggressively if they do occur. Some experimental models suggest that maintenance of cord perfusion pressure is beneficial. This is accomplished by adequate intravascular volume repletion and judicious use of pharmacological pressors. Treatment of accompanying systemic injuries should be carefully coordinated. A multidisciplinary approach facilitates injury management and transition to rehabilitation thereby attempting to minimize the emotional impact upon the patient.

Methylprednisolone in high dosage has shown sufficient sustained benefit in SCI to merit routine usage and has been the standard of care in the US since 1990. An initial bolus of 30 mg/kg over 15 minutes is followed by a 23-hour continuous infusion at 5.4 mg/kg per hour. Timing of drug administration is crucial; the bolus must be initiated within 8 hours of injury. New evidence has just been published from a double-blind prospective randomized trial which studyied tirilazad, a new aminosteroid with potent antioxidant effects, versus 48-hour methylprednisolone therapy as opposed to the traditional 24-hour methylprednisolone therapy. The group treated with solumedrol for 48 hours experienced significant improvements in motor function at 6 weeks and 6 months, particularly the group who received the initial bolus within 3 to 8 hours of injury. Those who received the initial bolus within 3 hours of injury showed similar recovery patterns with all three treatment regimens. Patients treated with tirilazad showed improvements intermediate to the two steroid patterns.

In the optimal management of spinal cord injury, high-dose methylprednisolone therapy is indicated. If the initial bolus is started within 3 hours of injury, 24-hour treatment is appropriate; if the bolus is not administered until 3–8 hours, treatment should be extended for a total of 48 hours. The therapy should not be given more than 8 hours after injury.

GABRIELLE F. MORRIS, MD
LAWRENCE F. MARSHALL, MD
San Diego, California

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Management of Internal Carotid Artery Occlusion

ACUTE OCCLUSION of the internal carotid artery produces a variety of clinical syndromes, including complete absence of symptoms, fluctuating neurological deficits, or various degrees of completed stroke. In the general population, the incidence of asymptomatic internal carotid artery occlusion in people older than 60 years has been estimated to be approximately 1%. Conversely, 27% of patients with occlusion documented by angiography were found to have a history of neurological symptoms, including transient ischemic attack (TIA) and stroke. In prospective studies, the reported annual rate of ipsilateral stroke in patients with asymptomatic internal carotid artery occlusion has been estimated at 4–5%, two thirds occurring ipsilateral to the occluded artery.

Strokes and TIAs after internal carotid artery occlusion have been attributed to four main pathogenic mechanisms: 1) emboli into the circle of Willis from the distal end of the occlusive thrombus; 2) emboli originating from a residual internal carotid artery "stump" through the external carotid artery; 3) emboli from ipsilateral common carotid artery, external carotid artery, or aorta through collateral channels; or 4) hypoperfusion distal to the occlusion. Because of the capacity of the cerebral circulation to develop collateral flow, chronic hypoperfusion after internal carotid artery occlusion is relatively uncommon. In this condition, autoregulation is impaired and cerebral blood flow is susceptible to changes in blood pressure or cardiac output. This concept led to the widespread application of extracranial-to-intracranial bypass procedures in the 1970s. Although a randomized, prospective trial showed no benefit from this bypass for preventing stroke in patients with internal carotid artery or middle cerebral artery occlusion, there may be a subset of such patients with truly impaired cerebral autoregulation who would benefit from surgical revascularization to augment flow. To identify this subgroup, cerebral vasodilating stimuli such as CO_2 or acetazolamide can be used to dilate the vascular bed transiently and determine the adequacy of cerebral hemodynamic reserve by transcranial Doppler monitoring or single-photon emission computed tomography (SPECT) imaging.

Distinguishing between high-grade internal carotid artery stenosis and chronic occlusion is critical, since the two conditions are treated in different ways. Whereas carotid endarterectomy has been shown to reduce stroke